

Highlights of this issue

BY SUKHWINDER S. SHERGILL

AGE, EXPERIENCE AND STIMULANT DRUGS IN SCHIZOPHRENIA

Schizophrenia is usually diagnosed during the critical period between adolescence and early adulthood. However, longitudinal epidemiological studies suggest that there may be earlier signs of an at-risk phenotype. In this issue, Tuulio-Henriksson *et al* (pp. 215–219) describe an association between verbal learning and memory deficits and an earlier age at onset of schizophrenia. Interestingly, IQ and working memory did not show this relationship. The authors suggest that neuropsychological testing in these areas is important in early-onset cases and that it may then be possible to tailor therapy towards these areas. The social contribution to the aetiology of psychosis is highlighted by the finding of an excess of victimisation experiences in people with psychosis, with many of these aversive experiences occurring before the onset of the disorder (Bebbington *et al*, pp. 220–226). The stress diathesis paradigm of schizophrenia is often used by patients and clinicians as part of an explanatory model, and these results suggest that this may have a role at the onset of the illness. More contemporary pharmacological models of psychosis include an updated role for dopamine. This sensitisation model can bridge the social aspect of victimisation experiences and learning deficits, as well as the antidopaminergic action of antipsychotic medication. Curran *et al* (pp. 196–204) review the literature on stimulant use and psychosis and find that even a single dose of a stimulant drug will exacerbate psychotic symptoms in patients. A lesser proportion of healthy subjects will also demonstrate psychotic symptoms in

exposure to stimulants, with the proportion rising with more chronic usage. It is unclear whether low-dose antipsychotic medication may have a role in preventing sensitisation. The role of endogenous stimulant release in response to stressful experiences poses further interesting research questions.

DEPRIVATION, DEPRESSION AND DAY HOSPITALS

Social disadvantage is associated with increased rates of psychological distress in adults. As there are continuities between psychological distress in childhood, adolescence and adulthood, one would anticipate similar risk factors in adolescence. However, a study by Stansfeld *et al* (pp. 233–238) of 11- to 14-year-olds in east London showed that there was little relationship between social disadvantage and psychological distress or depressive symptoms; indeed, Bangladeshi pupils, who were highly socially disadvantaged, demonstrated a lower risk of psychological distress than others. The authors propose that the presence of increased family support, religious belief or other ethnically related protective factors may play a role in explaining the latter findings. In a more affluent area, male patients attending their general practitioner (GP) in rural England were assessed for depressive symptoms. Shiels *et al* (pp. 239–244) report that patients with chest pain or low energy were more likely to be diagnosed with depression. However, there was little agreement between GPs and patients about the degree of depression. This follows the previously reported pattern of poor sensitivity in GPs' detection of depression. The effectiveness of acute psychiatric day hospitals is used by Burns

& Priebe (pp. 189–190) to illustrate the difficulties inherent in assessing the effects of an intervention at the service level but having the individual patient as the unit of outcome. They make the point in their editorial that there has been a vast increase in the amount of mental health services research, with most of this focused on the evaluation of innovative services – giving the impression that there is ongoing improvement, with outmoded services being replaced by more effective successors. However, they suggest that the reality is very different; one way forward may be to assess the community services currently available in the UK, and clarify the differences and commonalities between them in order to identify factors associated with sustainability, and to test out *a priori* hypotheses.

TEMPORAL INTEGRITY BUT PREFRONTAL DEFICITS

Both prefrontal and temporal cortex, including Heschl's gyrus, have been suggested to be abnormal in schizophrenia. A neuropathological examination of the latter region in patients with schizophrenia showed no differences compared with controls in the density of neuronal or glial cells. There was also no difference in patients with bipolar disorder or major depressive disorder. Cotter *et al* (pp. 258–259) reflect on their findings, suggesting that glial cell deficits may preferentially involve prefrontal cortex in affective disorders. An additional level of complexity is presented by Mendrek and colleagues (pp. 205–214), who demonstrate stable abnormalities in the functioning of the *left* prefrontal cortex during a working memory task in patients with schizophrenia, using functional magnetic resonance imaging. However, they found that activity in the *right* prefrontal cortex normalised with treatment with antipsychotics. They suggest that this underutilisation of the left dorsolateral prefrontal cortex may be a trait marker, whereas disturbance of the right dorsolateral prefrontal cortex may be a state-related phenomenon in schizophrenia.